



The origins of hand stiffness

Educational article - English edition

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Source article: Geoffroy C, Mespl   G. Les origines de la raideur de la main. Kin  sith  rapie, la Revue, 2020.

1. General introduction

Hand stiffness is the most frequent complication after injury, surgery or prolonged immobilisation. Once regarded as a simple mechanical restriction, it is a complex phenomenon involving biomechanics, tissue healing, collagen physiology, neuroplasticity, motor control and behavioural adaptation.

Geoffroy and Mespl   combine tissue science, clinical reasoning, neurophysiology and hand therapy. Stiffness rarely has one cause: functional limitation usually reflects interaction between oedema, adhesions, fibrosis, reduced muscle extensibility, bony impingement, sensory disturbance and disorganised movement patterns. This perspective changes assessment, prognosis and treatment.

2. General pathophysiology

Normal movement requires a precise balance between tissue gliding, skin extensibility, joint mobility, force transmission and sensorimotor control.

After injury, inflammation, oedema, fibroblast activation, collagen proliferation, disrupted mechanical loading and motor inhibition occur together. They change tissue mechanics, gliding, compliance and patient movement behaviour. Stiffness thus has local, systemic and neurological dimensions. The hand cannot be understood independently of the brain, sensory system and movement strategies.

3. Oedema-related stiffness

Oedema is probably the most frequent initial cause of lost mobility. The article uses Brand's analogy of a boat moored at low and high tide with a chain of unchanged length.

Without swelling, connective attachments between skin and fascia remain slack, permitting tissue mobility. Swelling separates the skin from deeper layers, tensions connective tissue and exhausts available skin slack. This immediately reduces finger flexion, skin gliding and joint motion.

Finger flexion requires approximately 12 mm of dorsal skin lengthening. Swelling increases this demand while skin mobility decreases, tissues become congested and compressive stresses rise.

Clinically, the end feel is soft, limitation relatively uniform and range sometimes varies with the trophic state. The authors prioritise oedema treatment before intensive mobilisation. Mobilising swollen tissue too aggressively may increase inflammation, fibrous organisation and fibrosis.



Proposed measures include drainage, compression, cryotherapy, cohesive bandaging, inflammation management, elevation and circulatory activation.

4. Skin-related stiffness

Palmar skin is thick, relatively inextensible, firmly attached, rich in mechanoreceptors and specialised for grasping. Dorsal skin is thin, mobile and extensible, accommodating finger flexion.

Even a small dorsal scar may therefore substantially restrict flexion and gliding and cause secondary adhesions. Scar adhesions restrict skin extensibility and extensor tendon gliding, with limited flexion or extension, pain and altered movement patterns.

Skin is a functional biomechanical component of digital movement. Treatment should address extensibility, gliding, relative movement between layers, sensory stimulation and adhesion prevention.

5. Collagen-related stiffness and cross-linking

The authors describe collagen maturation. Newly formed collagen is immature, disorganised and strongly influenced by mechanical loading. It progressively becomes organised mature collagen; the quality of maturation depends on loading, mobilisation, immobilisation and mechanical dose.

Cross-linking is central. Inappropriate loading-excessive tension, aggressive stretching, painful mobilisation or excessive immobilisation-may cause microinjury, restart inflammation and healing, increase fibroblast proliferation and favour abnormal collagen bridges.

Fibres lose their own mobility, interfibrillar movement and gliding, producing tissue stiffening. The article argues that mobilisation should remain within tissue elastic limits: excessive tension damages rather than reorganises tissue.

Conversely, gentle, prolonged, minimally painful and repeated mobilisation favours fibre orientation, remodelling and collagen maturation. Remodelling may remain possible for up to 18 months, with progressively decreasing adaptability.

6. Muscle-related stiffness

Prolonged immobilisation in a shortened position causes loss of muscle length, reduced compliance, sarcomere changes and reduced extensibility.

The limitation varies with adjacent joint position, overall muscle tension and functional chains. This variability may distinguish muscular from purely articular restriction and highlights the importance of muscle chains and whole-limb functional mobility.

Untreated muscle restriction may change joint loading, disrupt movement patterns, encourage compensation and maintain stiffness.



7. Bone-related stiffness and joint impingement

Bony blocks are among the most difficult forms. End feel is abrupt, hard, unmodifiable and independent of other joint positions. Causes include fractures, bony remodelling, subluxation, joint deformity and incongruence.

The authors describe a “pincer effect”: in interphalangeal joints, the functional axis lies anterior to the anatomical axis, producing anterior pinching and posterior opening. Increased bone volume after fracture may create cartilage overpressure, mechanical impingement and irreversible restriction. Rehabilitation alone is then insufficient and treatment is often surgical.

8. Movement patterns and neuroplasticity

Drawing on Roll’s work, the authors describe cortical hand-motor network changes after only five days of immobilisation: reduced cortical activation, altered motor maps and disorganised finger kinematics.

Consequences include loss of coordination, motor inhibition, functional non-use and compensatory strategies. Mechanical problems affect the brain, which can in turn maintain mechanical problems. Stiffness becomes neuromechanical, helping explain persistent pain, clumsiness, apprehension, loss of automatic movement and poor movement quality.

Proposed early stimulation includes proprioception, vibration, sensory stimulation, motor imagery and controlled active movement.

9. Clinical reasoning

End-feel assessment helps guide diagnostic reasoning.

Oedema-related stiffness: soft end feel, diffuse restriction and dependence on trophic condition.

Collagen-related stiffness: progressively firm end feel, potential improvement and dependence on tissue remodelling.

Bone-related stiffness: abrupt, unmodifiable end feel with early mechanical impingement.

Muscle-related stiffness: variation with joint position.

This differentiation informs technique, loading dose, orthotic selection, prognosis and surgical indications.

10. Early movement and mechanical dose

The traditional priorities are fear of stiffness and commitment to early movement. However, movement must be controlled, progressive, appropriate and minimally painful.

Overly aggressive mobilisation maintains inflammation, promotes microinjury and fibrosis and restarts pathological healing. Excessive immobilisation promotes oedema, disorganised collagen, muscle changes and cortical disturbances. The therapeutic challenge is precise mechanical dosing.



11. Implications for hand therapy

The article connects hand therapy with neurorehabilitation and tissue biomechanics. Treatment cannot be limited to passive range or aggressive stretching. It must incorporate inflammation management, gliding quality, sensory stimulation, motor control, neuroplasticity and mechanical load management.

This comprehensive approach supports individualisation, prevention and better-quality functional recovery.

12. Conclusion

Hand stiffness has biomechanical, histological, neurological and behavioural dimensions. Oedema, adhesions, cross-linking, reduced muscle extensibility, bony impingement and cortical disturbances interact continuously. This multidimensional perspective changes clinical reasoning, treatment choices and understanding of hand therapy.

